CONDUCTION DISTURBANCES IN HYPERTENSION

Dissertation Submitted to

THE TAMIL NADU DR. M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations
for the award of the degree of

M.D. BRANCH – I
GENERAL MEDICINE

GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI, INDIA

SEPTEMBER 2006

CERTIFICATE

This is to certify that the dissertation titled "**CONDUCTION DISTURBANCES IN HYPERTENSION**" is the bonafide original work of **DR. B. SURESH PRABU,** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in SEPTEMBER 2006. The Period of study was from August 2005 to April 2006..

PROF. S. NATARAJAN, M.D. Professor and Head of the Dept. of Medicine, Govt. Stanley Medical College and Hospital Chennai-600 001.	**PROF. A. K. GEETHA DEVI M.D.** Addl. Professor of medicine, Dept. of medicine, Govt. Stanley Medical College and Hospital, Chennai-600001

DEAN
Govt. Stanley Medical College & Hospital,
Chennai – 600 001.

DECLARATION

I, **DR. B.SURESH PRABU,** solemnly declare that dissertation titled **"CONDUCTION DISTURBANCES IN HYPERTENSION"** is a bonafide work done by me at Govt. Stanley Medical College and Hospital during 2005 under the guidance and supervision of my unit chief **PROF.A.K.GEETHA DEVI M.D**., Addl. Professor of Medicine.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

Place : Chennai.

Date :

(DR. B.SURESH PRABU)

ACKNOWLEDGEMENT

I owe my thanks to the Dean, Govt. Stanley Medical College and Hospital, **Dr. M.VASANTHA, MD.**, for allowing me to avail the facilities needed for my dissertation work.

I am grateful to **Prof. S. NATARAJAN, M.D.,** Professor and Head of the Department of Medicine, Govt. Stanley Medical College and Hospital for permitting me to do the study and for his encouragement.

I express my gratitude to **Prof. A.K.GEETHA DEVI, M.D.,** Addl. Professor of Medicine, Chief of Medical Unit IV, Govt. Stanley Medical College and Hospital for his valuable assistance and guidance.

.

I am extremely thankful to my Assistant Professors **Dr. NALINI KUMARAVELU, MD., and Dr. S. CHANDRASEKAR, MD.,** for their guidance and encouragement.

I am also thankful to my colleagues for their full cooperation in this study.

Last but not the least, my sincere thanks to all the patients who co-operated for this study without whom this study could not have been possible.

INTRODUCTION

Hypertension is one of the most common diseases of mankind which results in a very high incidence of morbidity and mortality.

Hypertension is the most important public problem of the modern world. It is one of the most common diseases which are easily detectable, effectively treatable and leading to fatal complication if left untreated.

The understanding of the pathophysiology of the hypertension has widened, still about 90 to 95% of the causes do not have an identifiable cause and is known as primary or essential hypertension. [40, 42] In small group of hypertension 5 to 10% an etiology could be identified, this is the secondary hypertension. According to JNC 7, hypertension is said to be present when the systolic and diastolic blood pressure exceeds 140 and 90 mm Hg respectively.

Hypertension causes an increase in left ventricular mass resulting in increased stiffness of the left ventricle leads to reduced coronary reserve [33,35,36,40,41], silent myocardial ischemia, and abnormal electrophysiological property of hypertrophied myocyte and conduction disturbances.

These changes probably accounts for the increased morbidity and mortality associated with hypertensive heart disease. Hypertension is also associated with increased cerebrovascular accidents [31, 32].

In the assessment of cardiac status in such patients ECG forms an important investigatory procedure and a very economical tool and easily applies to large number of population. Though normal ECG may be found in many cases of moderate to severe hypertension it still remains a useful means of diagnosis of hypertension associated with axis deviation, bundle branch block, fascicular block and myocardial infarction associated with hypertension [23,24,25,26].

AIMS

1) To study the incidence of conduction disturbances in patients with primary hypertension,

2) To identify the most common type of conduction defect and incidence among males and females.

3) The identify the role of diastolic blood pressure that will affect the prevalence of conduction disturbances.

REVIEW OF LITERATURE

ANATOMY OF THE CONDUCTION SYSTEM

The components of the conduction system are

a) S.A Node

b) Preferential inter nodal pathway

c) A.V Node

d) Bundle of HIS

e) Right bundle branch

f) Left bundle branch

g) Left anterior fascicles

h) Left posterior fascicle

THE SINUS NODE (SINO-ATRIAL NODE)

S.A Node is the pacemaker of the heart. It is a small flattened ellipsoid strip of specialized muscle about 3mm width, 15mm long and 1mm thick.

It is situated in the wall of the right atrium near the upper end of the sulcus terminalis and extending over the front of the opening of superior vena cava. The fibers of SA Node are 3-5 microns in diameter. In contrast to the diameter of 15-20 microns of the surrounding atrial muscle fibers.

INTERNODAL PATHWAY

These fibers connect SA Node to AV node through various pathways but most prominent are three pathways. These are as follows;

a) Anterior internodal tract of BACHMANN.

b) Middle internodal tract of WENCKEBACH.

c) Posterior internodal tract of THORAL.

THE ANTERIOR INTERNODAL TRACT

It leaves the anterior margin of SA Node, passes anterior to the superior vena cava to enter the crest of the A V Node.

THE MIDDLE INTERNODAL TRACT

Leaves the posterior margin of S.A node. passes posterior to the superior venacava, descends in the inter atrial septum and merges with the fibers of anterior internodal tract , to enter the crest of A.V Node.

THE POSTEROR INTERNODAL TRACT

It leaves the posterior margin of S.A Node, runs with the crista terminalis, curves through the valves of inferior venacava to enter the posterior margin of A.V Node.

THE ATRIO-VENTRICULAR NODE (A.V .NODE)

It is situated in the right atrial side of the interatrial septum just above the opening of the coronary sinus.

BUNDAL OF HIS

It passes from the A.V Node in the membranous part of the interventricular septum and divides into the right and left bundle branch, on either side of the septum.

RIGHT BUNDLE BRANCH

It travels down the septum to the anterior wall of the ventricle, enters the base of the anterior papillary muscle.

LEFT BUNDLE BRANCH BLOCK

It is a abroad band which usually divides into two divisions namely the left anterior fascicle and left posterior fascicle [1, 2]. These divisions showed are thought of as groups of interconnecting fascicles as opposed to individual, insulated, free running divisions.

The A V Node contains slow conduction fibers and is mainly responsible for the delay in the conduction of impulses from the atria to the ventricles, while the HIS Bundle and the trifascicular conduction systems are composed of rapidly conducting **HIS –PURKINJE** system [1,2,23,24,25.]

The Purkinje system consists of very large fibers, even larger then the normal ventricular fibers. The rapid transmission of impulse by purkinje system is probably caused by increased number of plexuses between the successive cardiac cells that make up the purkinje fibers. They have very few myofibrils and barely contract during the course of impulse conduction.

The terminal purkinje fibers penetrate about 1/3 rd of the the muscle mass and up to terminates the muscle fibers [23, 24, 25].

BLOOD SUPPLY OF CONDUCTION SYSTEM

S.A NODE ---- Right coronary artery

A.V NODE ---- Right coronary artery

A.V BUNDLE ---- Branch of Right coronary artery

BUNDLE BRANCH ---- Left coronary artery

Posterior fascicle alone has a blood supply from both coronary arteries [1, 2,23,24,25.]

INNERVATION OF THE HEART

It is by the cardiac plexus. This is supplied with both sympathetic and parasympathetic fibers. It is situated at the base of the heart and is divided in to a superficial and deep part, which is closely connected, several small ganglia are found in the plexus. The largest and most constant being the cardiac ganglion [1,2,25]. The superficial part of the cardiac plexus lies below the area of aorta, anterior to the right pulmonary artery.

The deep part of the cardiac plexus is situated in front of the bifurcation of the trachea, above the point of division of the pulmonary trunk and posterior to the aortic arch. The left coronary plexus gives branches to the left atrium and left ventricle.

Sympathetic fibers are concerned with cardiac acceleration and dilation of coronary arteries, while the parasympathetic fibers are concerned with slowing of the heart, rate and constriction of coronary arteries [1, 2, 23, 24]. The intrinsic cardiac nerve cells are limited to the atria and the interatrial septum. They are most numerous near the A V and S A nodes.

ELECTRO PHYSIOLOGY OF THE CONDUCTION SYSTEM

Along with nerve, skeletal muscle, and smooth muscle, heart muscle is one of the excitable tissues of the body [1,2,26]. It shares the biochemical properties with other excitable tissues but has unique electrical features as well. The electrical activity of the heart is responsible for coordinating the sequence of cardiac activation and contraction. Understanding the normal electrocardiogram (ECG) requires a substantial knowledge of normal cardiac electrophysiology.

THE SARCOLEMMA

The sarcolemma of cardiac cells is comprised of phospholipids bilayer with its associated membrane and fluid components. So that the membrane had been linked to a collection of protein icebergs floating in a lipid sea. The extrinsic protein often are glycocylated and provide structural support to the sarcolemma. The intrinsic protein serves as receptors to ion channels and pumps.

IONIC CHANNELS

Ionic channels are components of the membrane that permit movement of ions across the hydrophobic barrier of the cell membrane. These channels are thought to be membrane- spinning protein that contains water so that hydrated ions can cross the membrane. The channel selective to certain ions. Na+ channel preferentially conducts Na+ and is less permeable to K+ or Ca2+ ions. Also channels have gating mechanism i.e, they may be open or closed depending on

the chemicals(agonists)in the channel or the transmembrane voltage field, the degree to which the is open may vary once open. Channels have characteristic open times, 1ms for Na+ channels and more than 100 ms for Ca2+ channels. Each channels seem to operate independently i.e., the probability that a channel will be open or close is not influenced by the state of neighboring channels.

Three different membrane ion channels play important roles in causing voltage changes of action potentials in the cardiac muscle.

1) Fast **SODIUM c**hannels

2) Slow **CALCIUM-SODIUM** channels

3) **POTASSIUM** channels.

RESTING TRANSMEMBRANE VOLTAGE

Cardiac cells have a large transmembrane voltage difference during diastole, about -60 to -90 mv relative to the extra cellular fluid potential. This resting tran smembrane voltage or potential is an important factor in the electrical behavior of the cell.e.g, determining the action potential and also in regulating the trans membrane ion transport. In normal cardiac muscle the resting transmembrane concentration gradients for ions such as K+ and Na+ are established by active ionic pumping and the membrane conductance for those ions.

THE SODIUM PUMP

There is significant resting Na+ influx in cardiac cells. If Na+ ions were not extruded by the cell, the resting potential would decrease as Na+ ions were accumulated. Extrusion of Na+ from the cell requires energy because both electrical and chemical gradients oppose removal of Na+. The energy for Na+ pumping is provided by the alpha subunit of membrane associated Na-K adenosine triphosphatase (ATP ase) that extrudes three Na+ and pumps in two K+ for each molecule of ATP that is hydrolysed . This enzyme is stimulated by catecholamines and inhibited by digitalis glycosides. The activity of the Na+ pump is electrogenic, i.e,more positive change is pumped out than pumped in (3:2 Na+-K+ ratio)[1,2.] Under resting conditions the pump current makes contribution to resting membrane potential. Under conditions of increased Na+ entry, (e.g., increased heart rate), pump current will contribute more to diastolic membrane potential driving to more negative values.

THE CARDIAC ACTION POTENTIAL

Like other excitable cells cardiac cells produce an action potential when activated. Of all the excitable cells in cardiac cells have the longest action potentials their repolarization is the slowest. The cardiac Purkinje fiber action potentials has FIVE phases

PHASES:

0- Rapid depolarization

1- Immediate rapid repolarization

2- Slow repolarization or plateau

3- Rapid repolarization

4- Diastolic interval.

Other cardiac cell types have distinctive action potential contours that differ markedly from that of the purkinje fibers. They can be thought of as belonging to one of two major groups, fast or slow action potentials.

FAST ACTION POTENTIALS AND FAST CONDUCTION

Most cardiac cells, such as ordinary or specialized atrial fibers, purkinje fibers, and ventricular muscle cells, have fast action potentials. These cells have high resting membrane potentials, and when activated, generate a fast-rising, large amplitude phase 0. This type of phase 0 is associated with very rapid conduction cells with fast action potentials tend to have completed and highly developed intercellular connections. The large surface area of complex cellular junctions provide a low resistance pathway for current most highly developed in purkinje fibers ,and conduction is most rapid in these fibers.

Fast action potential in the heart is generated by an inward rush of Na+ through an ionic channel that is selectively permeable to Na+ ions when the cells are activated. The fast channels are activated when membrene potential is rapidly

brought from it resting valve of -90 mv to the threshold voltage, about -75mv. The Na+ channel is activated quickly, inactivated quickly (time constant 0.5 to 2 ms) and has a very high value for maximum ionic conductance when fully activated. The Na+ channel is only open for 1 ms to 2 ms, but the inward Na+ current is intense during that moment. The main modulator of Na+ channels conductance is the value of resting membrane potential decreases and becomes less and less negative ,the Na+ channels will inactivate more and more so that the inward Na+ current during activation become less and less intensive .The weaker in the Na+ current ,the smaller the amplitude and rate of rise of phase 0 and the slower impulse conduction will be at a resting membrane potential of about -60 mv ,the Na+ channels is totally inactivated and no response can be elicited even with very strong stimulus. When the Na+ channels activates from a normal resting membrane potentials the intense potential to very positive valves (+30mv) ,When the channel inactivates rapidly and will remain inactivated and incapable of responding to stimuli until the cell repolarizes, the Na+ channel reactivates progressively and reaches its maximum at membrane potential near -90 mv [1,2] . When the cell has to polarize to -65 mv or so, it can be activated again, however stimulation at this low value of membrane potential gives rise to very low amplitude, slow-rising action potential. As repolarization proceeds from -65mv to -85mv, response to premature stimulation will yield action potentials with larger amplitude and faster depolarization rates.

This voltage dependent responsiveness of fast action potentials is an important factor in arrhythmogenesis and operates in abnormal parts of the heart,

where cells are depolarized for intense, by local hyperkalemia, by stretch injury or during premature activation. The Na+ channels are blocked by TTX and many anti arrhythmic drugs with class I action but not by Ca2+ channel blocking drugs.

SLOW ACTION POTENTIALS AND SLOW CONDUCTION.

The action potentials of sinus node P cells and atrioventricular (A.V) nodes N cells are very characteristic and quite similar. These cells have a low maximum diastolic value of membrane potential and a small amplitude and relatively slow rising upstroke ,i.e. phase 0, when they are activate potentials are associated with very slow conduction.

So, there are anatomic reasons for the slow conduction in the sinus node and A V node. The cells to cell connection between sinus node P cells and A V node N cells and their neighbors are sparse and primitive, contributing to the slow spread of excitation in these structures.

The ionic basis for low potentials in anionic channel that, when activated, is selectively permissible to calcium (Ca2+) and to a lesser extent. To sodium ions, the channels that carries slow inward current activates at rather positive valves of membrane potential such a -40 mv to -50 mv .The slow or L type Ca2+ channel activates (open) slowly, inactivates slowly (time constant 50 ms to 100 ms) and has a low valves for maximum ionic conductance. When fully activated. The conductance of the channel is regulated to some extent by resting membrane potential and increased substantially by catecholamine. The L type Ca2+

channel can be blocked by manganese or by drugs such as verapamil, diltiazem or nifedipine.

The slow propagation in the sinus and A V node permits reentrant excitation to occur in very small areas despite the long refractory period found in cardiac muscle.

SLOW ACTION POTENTIALS IN FAST FIBERS

Under abnormal circumstances, fibers that normally give rise to fast action potentials can develop slow action potentials for example, purkinje fibers, the prime example of a fast fiber type can develop slow action potentials under a variety of conditions, increasing the extra cellular K+ concentration to above 16 meq/lit will reduce the resting membrane potential of a purkinje fibers, the threshold for the Ca2+ channels will shift in a negative direction and the maximum inward Ca2+ current obtained during activation will increase under these circumstances, electrical stimulation of the purkinje fiber will evoke a slow responses are unaffected by Na+ channels blocking agent such a TTX but are abolished by Ca 2+ channel blocking agents. Action potential of actually ischemic cells resembles the slow responses that occur in high K+, high catecholamine conductions.

Under these conditions, the slow depolarization probably is caused by depressed Na+ channel action potentials. Ischemic slow action potentials are abolished by selective Na+ channel blockers such as TTX but not by Ca2+ channel

blockers. Purkinje fibers can generate slow action potentials during a variety of abnormal condition via a variety of ionic mechanisms.

REPOLARIZATION OF CARDIAC ACTION POTENTIALS

One of the most striking attributes of the cardiac action potential is its long plateau, membrane potential remains more positive than -50 mv for several hundred milliseconds. This contrast with the brief action potentials of peripheral nerves and skeletal muscle fibers which typically last for less than 5ms.The long plateau provides adequate Ca2+ for contraction and prevents very rapid heart rates. Several ionic mechanisms are known to contribute to the long plateau.

First, K+channels in purkinje fibers show inward going rectification positive to -50 mv so that the outward repolarization currents carried by K+ decrease during the plateau of the action potential. Second, the cell tends to be held at plateau voltage by inward current carried by Na+ and Ca2+. The slow or secondary inward current normally is triggered by the depolarization caused by phase 0 of the action potential, during the plateau, the small inward and small outward currents are almost perfectly balanced so that membrane potential changes very little for 200 ms to 400 ms. phase 3 of the action potential is due to inactivation of iCa –L and activation of I_k, an outward current. As the slow channel inactivate the iCa-L decreases and the cell tends to repolarize.

The K+ accumulate in narrow extra cellular cleft may play a role in repolarization and certainly complicates attempts to study repolarizing K+ currents in multicellular preparation with voltage clammy techniques.

The normal heart beat is governed by a specialized system that spontaneously generates and distributes each impulse though the heart in a coordinated way. Normally, spontaneous impulse generation is much faster in the sinus node (60 to 100 beats/mints) than in the sinus nodes in control of cardiac rhythm.

VELOCITY OF CONDUCTION IN CARDIAC TISSUE

TISSUE	CONDUCTION RATE(m/sec)
S-A node	0.05
Atrial pathways	1
A-V node	0.05
Bundle of HIS	1
Parkinje system	4
Ventricular muscles	1

TIME TAKEN FOR IMPULSE TO TRAVEL

TRAVELING PATHWAY	**TIME (seconds)**
S-A node to A-V junctonal fiber	0.04
Junctional fibers to A-V bundle	0.11
A-V Bundle to purkinje termination	0.03
Purkinje to cardiac muscle	0.03

CONDUCTION DISTURBANCE --- BASIC PRINCIPLES

Among **various** forms of intraventricular block, bundle branch block is most common and best recognized.

The various intraventricular conduction disturbances are…

1) Right bundle branch block
2) Left bundle branch block
3) Left Anterior fascicular block
4) Left posterior fascicular block
5) Bifacicular block
6) Trifacicular block
7) Non specific intra ventricular conduction disturbance.

BASIC PRINCIPLES

If one of the branches of the bundle of HIS is blocked by disease, the impulse travels down to the other ventricle first having activated this ventricle the impulse spreads through the septum to the ventricle on the side of the block and in turn activates it. Thus the ventricles are activated one after the other instead of simultaneous activation. This results in prolongation of the QRS interval and S-T segment slopes off in the direction opposite to the main QRS deflection in cases of bundle branch black.

The incidences of RBBB and LBBB are equal in the Framingham study. In patients with evidence of cardio vascular disease most of the (Am. J.Card 1981; 47,931) patients who developed LBBB had antecedent or shortly to appear Hypertension, cardiomyopathy or symptomatic ischemic heart disease, [40, 42, 46].

Bundle branch block can be produced by involvement of the fibers in the branch or in the HIS bundle itself by many pathological processes. Very commonly it is due to either ischemia or fibrosis associated with ventricular hypertrophy as in systemic hypertension.

Same conditions can also be responsible for other types of intraventricular conduction disturbances in the form of fascicular block. In most patients, unifasciculsr, bifascicular or trifascicular disease is secondary to readily apparent organic heart disease [46.] This is associated with increased fibrosis with secondary involvement of the Trifascicular conduction system.

Histologicaly in hypertensive and arteriosclerotic heart disease, moderate fibrosis, fatty infiltration and arteriosclerosis were present in the approaches to the A V node in some cases.

In other cases there were complete fibro elastic disruption of right bundle branch. The penetrating bundle with the seat of moderate fibrosis, while the branching bundle showed marked fibrosis in few cases.

Some intra ventricular conduction disturbances are due to a diffuse conduction delay during the entire ventricular depolarization rather than because of a localized block at one of the bundle branches. The QRS configuration in such cases will be an exaggeration of the underlying QRS complex. This type of defect is known as diffuse or non specific intraventricular conduction defect. It superficially resembles LBBB but lacks its characteristic features. Some times encountered in elderly even with out significant heart diseases.

Bundle branch blocks are recognized by their prolongation of the QRS interval [23,24,25,29]. While some defects like the anterior or posterior fascicular block are associated with normal QRS interval. LBBB tends to produce more widening of QRS than RBBB.

Bundle branch block is commonly fixed and permanent but it may occur intermittently. Intermittent Bundle branch block may either depend upon heart rate or independent of heart rate [23, 24].

When the conduction defect involves both the right and left branches “Bilateral bundle branch block” occurs which may present as one of the many forms

of Bundle branch block depending up on which part of the conduction system is involved [23,24,25.]

Complete AV block is produced when the block in both the bundle branches is complete. Bundle branch block can be encountered in normal as well as in diseased heart but the presences of left bundle block nearly always indicate a diseased heart.

DIAGNOSTIC CRITERIA OF CONDUCTION DEFECT

DIAGNOSTIC CRITERIA OF "RIGHT BUNDLE BRANCH BLOCK" (RBBB)

It results from conduction delay in any portion of the right sided intraventricular conduction. The delay occurs in the main right bundle branch itself, in the branch of HIS or in the distal right ventricular conduction.

RBBB is also a variety of Unifascicular block. However in this Unifascicular block impulses are delivered by the left bundle branch and then must cross the septum to activate the right ventricle. This produces QRS widening. Complete RBBB is characterized by a wide QRS of >0.12 sec. The essential electrocardiograpic features of RBBB are,

1) QRS complex 120 msec or greater
2) Broad, notched R' waves (rsr', rsR') in right precordial leads. (V1and V2)
3) Wide and deep S wave in left precardial leads (V5 and V6).
4) Secondary S-T and T wave changes in V1 to V3.

CLINICAL SIGNIFICANCE

It is a common finding in general population and many patients with RBBB have no clinical evidence of structural heart disease [1, 2, 23, 24].

However new onset RBBB does produce a higher rate of coronary artery disease, congestive heart failure and cardiovascular mortality.

Brugada syndrome has been described in which a RBBB like pattern with persistent S-T segment elevation in right precordial leadsand its is associated with susceptibility to ventricular tachyarrhythmia and sudden cardiac death.

The diagnosis of RVH is more difficult when it is assocoated with RBBB because of the accentuated positive potential in lead V1, RVH is suggested [1, 23,24,25,26.]

LEFT BUNDLE BRANCH BLOCK

In this condition the left ventricle is activated after the right ventricle and initial septal action begin from right to left instead of the normal left to right.

It is due to conduction delay or block in any of several sites in the intraventricular conduction system, including the main left bundle branch ,in each of the two fascicles or less commonly within the fibers of the bundle of HIS that become the main left bundle branch block.

DIAGNOSTIC CRITERIA.

1) QRS duration 120 msec or greater
2) Broad, notched R waves in lateral precordial leads (V5 and V6) and usually Lead I and aVL.
3) Small or absent initial ‘r’ waves in right precordial leads (V1 and V2) followed by deep S waves.
4) Absent septal q waves in Left sided leads.
5) Prolonged intrinsicoid deflection (> 60 msec in V5and V6).

CLINICAL SIGNIFICANCE

The presence of LBBB almost always indicates a disease of heart [1, 2, 24, 25,] particularly coronary and hypertensive heart disease, less commonly it may be encountered in cardiomyopathy, myocarditis, and congenital heart disease.

LBBB in an apparently healthy heart is extremely unusual. It is more common than RBBB in elderly individuals with diseased heart. It is associated with reduced long term survival and with 10 yrs survival rates as low as 50%. It reflects the severity of the underlying cardiac disease.

The duration of the QRS complex in LBBB correlate **inversly** with left ventricular ejection fraction [1,23,23,24].

FASCICULAR BLOCKS

Under normal conditions activation of left ventricle begins simultaneously at the insertion sites of the fascicles. It results in activation of these sites sequently rather than simultaneously.

LEFT ANTERIOR FASCICULAR BLOCK

Since the impulse conducted via the posterior division is directed superiorly and to the left, a marked left axis deviation is observed in LAFB.

DIAGNOSTIC CRITERIA

1) A narrow QRS complex --- Less then or equal to 120 msec
2) Left axis deviation --- mean QRS axis -45 to -90.
3) rS pattern in the leads II ,III and aVF.
4) qR pattern in lead aVL

CLINICAL SIGNIFICANCE.

LAFB is common in person with overt cardiac disease. It has minimal or no independent prognostic significance. It is associated with cardiac and systemic condition like MI, especially occlusion of the left anterior descending coronary artery, LVH, hyper tropic and dilated cardiomyopathy and degenerative disease [1, 2,23,24,25.]

The development of LAFB with rS complexes in II, III and aVF can mask the Q wave of a prior inferior myocardial infarction.

LEFT POSTERIOR FASICULAR BLOCK

Conversely in LPFP, left ventricle is activated via intact anterior division. Since the impulse conducted through the anterior division is directed inferiorly and to the right, right axis deviation is produced.

DIAGNOSTIC CRITERIA

1) Frontal plane mean QRS axis less than 120 degrees
2) RS pattern in leads I and aVL with qR patterns in inferior leads.
3) QRS duration of less than 120 msec

CLINICAL SIGNIFICANCE.

This is rare to occurring in a healthy individual. The incidence of left posterior hemi block is much lower than LAFB [1, 2, 23, 25]. The possible explanation being the posterior division is shorter and thicker than and less influenced by the

stresses of out flow pressure, because of its inflow tract structure It also have a double blood supply compared to the anterior fascicle.

BIFASCICULAR BLOCK

It refers to the block of two fascicles of the trifascicuiar conduction system. The combination producing bifacicular blocks are,

1) Pre divisional block of LBBB

2) RBBB with LAFB

3) RBBB with LPFB

RBBB with LAFB is diagnosed when V1demonstrate RBBB and frontal plane leads demonstrate Left axis deviation.

SYSTEMIC HYPERTENSION

DEFINITION

It is a disorder of circulatory regulation and continuous variable and whatever number is used to define Hypertension will be Arbitary. The lower limit for the definition of hypertension has changed from 160/95 to 140/90 mm hg.

It is another way to expressed by lateral force exerted by the blood column per unit area of the vascular wall that is expressed in mm Hg.

JNC-7 CLASSIFICATION FOR HYPERTENTION

BP Category	BP Range(sys,dias in mm hg)	Lifestyle modification	Drugs
Normal	**<120 and <80**	**Encouraged**	**Not indicated**
prehypertension	**120-139 or 80-89**	**Recommended**	**Not indicated**
Stage-I	**140-159 or90-99**	**Recommended**	**Single drug**
Stage-II	**>160 or>100**	**Recommended**	**Double drugs**

Threshold Values for NORMAL versus ABNORMAL Blood Pressure (mm Hg)

Source	Office readings	Home Readings	Ambulatory BP
Jnc-7	140/90		
Tsuji et al.	140/90	137/84	
De Guademaris et al.	140/90	127/83	
Pickering	140/90	135/85	135/85
Staessen et al.	140/90		133/82

HISTORY OF HYPERTENSION

YEAR	EVENTS
1773	Stephen Hales measured BP for the first time in HORSES by measuring height of a column of blood in a vertical tube inserted in to CRURAL artery.
1895	Riva Rocci invented sphygmomanometer
1896	Sir, Clifford Albert- made distinction between Hypertension due to Renal disease and Hypertension in which no renal pathology can be found.
1898	Tigersteds and Bergman isolated rennin from renal cortex
1904	Amleard and Beayard studied Hypertension patients and observed a positive chloride balance associated with arise in Blood pressure.
1915	M.C Korokoff, Russian surgeon introduced Auscultatory method of recording Blood pressure.

CAUSE OF HYPERTENTION

I .SYSTOLIC and DIASTOLIC

A) PRIMARY/ESSENTIAL or IDIOPATHIC

It is idiopathic but it believed that arises as a result of multilateral condition, factors that produce hypertension.

GENETIC FACTORS

MONOGENIC FORMS

a) Glucocorticoid – remediable aldosteronism

b) Liddle's Syndrome.

POLYGENIC FORMS

a) Angiotensinogen gene

b) Na+-Li + Counter transport

c) Epithelial amiloride – sensitive sodium channel

d) Nitric oxide generation

e) Alpha- adducin defect

f) G beta 3 subunit

g) Insertion /deletion of ACE gene

B) SECONDARY

1) RENAL CAUSE

A) RENAL PARENCHYMAL DISEASE

1) Acute glomerulonephritis

2) Chronic Nephritis

3) Polycystic disease

4) Diabetic Nephropathy

5) Hydronephrosis

B) RENOVASCULAR CAUSE

1) Renal artery stenosis

2) Intrarenal vasculitis

C) RENIN PRODUCING TUMOURS

D) PRIMARY SODIAM RETENTION

Liddle's and Gordon's syndrome

2) ENDOCRE

a)Acromegaly

b) Hypothyroidisum

c) Hyperthyroidism

d) Hypercalcemia

e) Adrenal

1) Cortical

i) cushing's syndrome

ii) Primary aldronism

iii) Congenital adrenal hyperplasi

iv) Apparent mineralocorticoid excess (licorice)

2) Medulary cause -- pheochromocytoma

f) External chromatin producing tumours

g) Carcinoid

h) Exogenous hormones

1) Estrogen

2) Glucocorticoids

3) Mineralocorticoids

4) Sympathomimetics

5) Tyramine containing foods and

6) Monoamine oxide inhibitors

3) COARCTATION OF AORTA

4) PREGNANCY INDUCED HYPERTENTION

5) SLEEP APNEA

6) NEUROLOGICAL DISORDER

a).Increased intracranial pressure

1) Brain tumor
2) Encephalitis
3) Respiratory acidosis
4) Quadriplegia
5) Acute porphyria
6) Familial dysautonomia
7) Lead poisoning
8) Guillain – barre syndrome

b)ACUTE STRESS (INCLDING SURGERY)

1) Psychogenic hyperventilation
2) Hypoglycemia
3) Burns
4) Pancreatitis
5) Alcohol withdrawal
6) Sickle cell crisis
7) Post resuscitation
8) Post operative
9) Increased intravascular volume
10) Alcohol and drug use

II) SYSTOLIC HYPERTENSION

A) INCREASED CARDIAC OUTPUT

1) Aortic valvular regurgitation
2) Aortoventricular fistula , patent ductus arteriosus
3) Thyrotoxicosis
4) Paget ‘s disease of bone
5) Beriberi
6) Hyperkinetic circulation

B) RIGIDITY OF AORTA

III) IATROGENIC HYPERTENSION

A) DRUGS

7) Non steroidal anti –inflammatory drugs
8) Sympathomimetic amines
9) Estrogen and estrogen analogs
10) Corticosteroids
11) Methylxanhtines
12) Cyclosporine’s
13) Erythropoietin
14) Cocaine
15) Nicotine
16) Phencyclidine
17) Herbal ecstasy

18) With drawal from certain Drugs

a) Beta blocker

b) Alpha agonist

c) Opioids

d) Ethanol

e) Calcium antagonist

PREVALENCE OF HYPERTENSION

AGE (IN YEARES)	HYPERTENSIVES %
18-29	4
30-39	11
40-49	21
50-59	44
60-69	54
70-79	64
>80	65

FACTORS AFFECTING HYPERTENSION

1) VASCULAR RESISTANCE
2) ENDOTHELIAL CELL FUNCTION

   Affected by; a) blood viscosity

   b) Vascular wall stem condition

   c) Blood flow velocity

   d) Arterial wall thickness

EARLY HYPERTENSION

It is mainly due to increased Cardiac output with out change of peripheral resistance.

FACTOR MEDIATED ARE:

1) Increased tissue demand or hypervolemia
2) Cardiac stimulation as by adrenergic hyperactivity
3) Slight hypokalemia with hypercalcaemia
4) Na+-Ca2+ exchange mechanism.

CHRONIC ESTABLISHED HYPERTENSION

Here the Cardiac output was normal or reduced variable mechanism

1) increased vascular resistance
2) decreased diameter of arteriolar lumen

3) Increase vasoactive factors enhance reactivity of smooth muscle cells or structural change in vessel wall.

THEORIES TO EXPLAIN THE CHANGES

1) THEORY OF AUTO REGULATION

Initial high Cardiac output cause over perfusion of tissues and secondary rise in systemic vascular resistance to counter balance the increase in flow, this restore the cardiac output to normal valve.

(Guyton et al, Leeding ham, Pelling)

2) PRIMARY PATHOLOGICAL CHANGE IN PERIPHERAL VASCULATURE OR VASCULAR REMODELLING THEORY

a) Hypertrophy of smooth muscles of resistance vessels in which vascular endothelium play an important role by its release of growth factors and biochemical mediators,

b) These changes causes increased vascular resistance.

3) GENETIC THEORY

a) Primary hypertension is a polygenic trait, sharing strong dependence on multiple environmental factor as well as heterogeneity of genes.

b) ACE gene, Angiotensinogen, AT receptor gene is also responsible.

c) Salt sensitivity and volume factors may play an important role in pathogenesis.

ISOLATED SYSTOLIC HYPERTENSION

In this entity the systolic blood pressure then 140 mm hg with diastolic blood pressure of less than 90 mm hg 1, 2, 40, 42.

CAUSES ARE:

a) Remediable functional causes like

-- Increased stroke volume and increased adrenergic activity.

b) Non- remediable structural cause

-- Athererosclerotic changes of vascular wall

During therapy improvement of large arterial compliance should be a goal because systolic blood pressure is an important determinant of myocardial wall stress.

HYPERTENSIVE HEART DISEASE

Heart is one of the organs most affected as a consequence of direct and indirect effects of hypertension. Direct effects account for the presence of Left ventricular hypertrophy and the resultant hypertensive disease [33, 34,40,41,42.] It is an

adaptive mechanism of the heart of the systolic overload of the left ventricle due to increased peripheral vascular resistance.

Left ventricular hypertrophy is defined as an increase in the muscle mass of left ventricle. The increases in muscle mass lower the coronary reserve and enhance the cardiac oxygen requirement. LVH is an independent risk factor for sudden death for myocardial death, congestive heart failure and other cardiovascular morbidity. The Framingham heart study has documented that the risk of these complications increases six to eight fold with the occurrence of Left ventricular hypertrophy.

MORPHOLOGICAL PATTERNS OF LVH

The classic form of **CONCENTRIC HYPERTROPHY** is defined as a thickening of the septum and posterior wall of the left ventricular at the expense of the chamber volume.

ECCENTRIC HYPERTROPHY is the thickening of the chamber wall with concomitant chamber dilatation. It occurs in the late phase of hypertensive heart disease a precursor of congestive heart failure.

Pathological cardiac hypertrophy may be associated with adverse effects on cardiac muscle including..

1) Altered diastolic tension
2) Increased college content

3) Alterations in Myocardial perfusion
4) Abnormal electrophysiological properties
5) Decrease contractility

RENIN-ANGIOTENSIN SYSTEM –IT'S ROLE IN LVH

It is suggested that chronic activation of Renin-angiotensin system and, ventricular loading is associated with the development of myocardial fibrosis in the Right and Left ventricle there appears irrespective of myocyte hypertrophy or necrosis.

In this regard the role of Renin-angiotensin, bradykinin, prosotaglandin system in regulation of myocardial collagen metabolism is well documented [34,44,45,46].

MYOCARDIAL COLAGEN MATRIX

The major structural proteins of the myocardial collagen matrix are the fibrillar collagen type I and type II. The concentration of type I determine stiffness of the myocardium. Myocardial fibroblasts contain the mRNA for type I and III collagens. An increased level of mRNA and collagen synthesis occurs in the myocardium in response to the Angiotensin II stimulation of fibroblast. While prostaglandin E2 inhibit the collagen synthesis in the fibroblast, collagen degradation is mainly effected through the enzyme MMP-1(matrix metallo protein), a key enzyme in collagen degradation.

ANGIOTENSIN II

It appears to be responsible for the disproportionate and diffuse accumulation of fibrillar collagen within the cardiac interstitium in hypertensive heart disease. while PG E2 counteracts the adverse tropic effects of Angiotensin II on myocardial collagen matrix.

ANGIOTENSIN RECEPTORS

The direct effect of Angiotensin II on cardiac fibroblasts require the presence of specific receptors on these cells .The presence of such Angiotensin II receptors type I and type II on cardiac fibroblasts and human myocardium has been shown.

AT-1 receptors is predominantly involved in the mediation of collagen synthesis whereas MMP-I inhibition by Angiotensin II may be mediated through AT-2 receptor. Other factors that may be involved in the myocardial remodeling in hypertensive heart disease could be the Insulin like growth factor I, receptors to catecholamine which may decrease the fibroblast mediated collagen synthesis.

These findings suggest that a direct interaction between Angiotensin –II and PGE 2 and cardiac fibroblast could be of major importance in requiring myocardial fibrosis. Chronic inhibition of cardiac angiotensin converting enzyme would induce both inhibition of myocardial angiotensin II generation and elevation of myocardial PGE2 levels.

This may explain why the ACE inhibitors Lisinopril has been shown to be a powerful agent in regressing myocardial fibrosis in hypertensive heart disease.

INVOLVEMENT OF CORONARY CIRCULATION

Patient with hypertrophied ventricles often exhibit signs and symptoms of myocardial ischemia in the absence of coronary artery obstructive disease. This has been proved by many angiographic studies in hypertensive patients who complain of anginal pain. These patients often demonstrate abnormal finding in exercise electrocardiography and myocardial Thallium 201 scan and abnormal lactate metabolism during pacing, suggestive of myocardial ischemia.

Studies in animals and men indicate that the major conduit epicardial coronary vessels are enlarged in hypertrophied ventricles. This is however less compared to the amount of increase in ventricular mass. This may result in a minor increase in total coronary resistance.

The effect of hypertrophy on smaller coronary arterial vessels remains controversial.Although some qualitative studies have suggested various abnormalities, an extensive quantitative studies have suggested various abnormalities an extensive quantitative study on dogs by Tomanek et al .has not demonstrated any consistent adverse alteration in resistant vessels. No significant alterations in wall to lumen ratios, were observed in coronary arterial vessels over a large range of sizes even though physiological measurements reveled significant increase in minimal coronary vascular resistance.

CAPILLARY DENSITY

Many studies have demonstrated a decrease in capillary density in the subendocardial layers of hypertrophied ventricles secondary to pressure overload [33,35,36,40]. This may result in increased diffusion distance, the significance of this to be defined by further study.

The physiological function of coronary collateral circulation is not enhanced by the pressure induced cardiac hypertrophy.

EFFECT OF LVH ON CORONARY RESERVE

Both volume and pressure induced hypertrophy in patients of all ages are associated with moderately decreased coronary reserve [33, 35,40,41,42]. This has been readily demonstrated in various studies.

The impairment of coronary reserve in arterial hypertension is mainly based on structural and functional alteration in the coronary resistance vessels [41, 42]. Myocardial hypertrophy also increases the extravascular compressive forces that elevate the extra vascular component of coronary resistance due to myocyte hypertrophy and interstitial and perivascular fibrosis.

Coronary reserve may also be impaired due to increased metabolic demands under baseline conditions. These changes result in an abnormal perfusion of the hypertrophied ventricles and it severe enough may lead to intermittent episode of severe subendocardial ischemia and eventually to diffuse sub endocardial fibrosis.

Recent electrophysiological studies by Martins et al have demonstrated that in the presence of myocardial ischemia conduction abnormalities and rapid ventricular tachycardia develop in hypertrophied ventricles.

This view is supported by the findings of a recent study of electrophysiology in hypertensives which has suggested that the increased electrophysiological disturbances may be related to an increased amount of fibrous tissue or altered collagen content.

PREVALANCE OF LVH

Data from Framingham cohort has demonstrated a prevalence of LVH of 16% for men and 19% for women. After the age of 70 yrs it is 30% for men and 49% for women [43.]

Echocardiographic data from Hammond et al have shown an overall LVH prevalence of 20% for patients with mild hypertension, with more severe hypertension,the prevalence may reach 50%..

DETERMINENT OF LVH

AGE: A recent multi centre trial confirmed that there was no association of age with wall thickness or left ventricular mass after adjustment for BP and body mass index.

SEX: Women have smaller LV mass than men for any given level of arterial pressure.

RACE: Black race is more prone for hypertensive complication than white race.

OBESITY: Framingham heart study has demonstrated a NINE to TEN fold increase in prevalence of LVH depending on the grade of obesity.

SALT INTAKE: Number of studies has shown that salt intake is a strong determinant of LVH independent of blood pressure.

ALCOHOL INTAKE: Relationship of alcohol intake and hypertension is significant as indicated by the intersalt study, but the association of alcohol with less clear.

LVH has been indicated as a significant and most powerful risk factor for future cardiovascular events causing morbidity and mortality. LVH and its sequence can be reduced by specific antihypertensive therapy, Future epidemiological studies are necessary to document the clinical benefit of a reduction in LVH.

ECG CRITERIA TO DIAGNOSE LEFT VENTRICULAR HYPERTROPHY

Sokolow-lyon index --- Sv1+(Rv5orv6) > 3.5 mv

R aVL>1.1mv

ROMHILT-ESTES POINT SCORE SYSTEM.

1. Any limb lead R wave or S wave > 2mv ---- 3 Points

   Sv1or Sv2 >3mv (30 mm)

   Rv5 or Rv6 > 3mv (30 mm)

2. ST-T Wave abnormality (no digitalis) ---- 3Points

   ST-T Wave Change with digitalis treatment ---- 1 Point

3. Left atrial abnormality ---- 3 Points

4. Left axis deviation > -30 degrees ---- 2 Points

5. QRS Duration > 90 Msec ---- 1 Point

6. Interensicoid deflexon V5 or V6 > 50 Msec ---- 1 Point

CORNAL Voltage Criteria ------ Sv3 + RaVL >2.8 mv(men)

Sv3 + RaVL > 2.0 mv(women)

CORNAL Voltage equation ------ Risk of LVH = $1/ (1+ e^{-exp)}$

Exp = 4.458 – 0.092 (RaVL + SV3) – 0.306 TV1 -0.212 QRS-278 PTF V1 -0.859 (sex)

NOVACODE Criterion (men)

LVMI9gem/m2= -36.4 +0.01 RV5 + 0.2 SV1 + 0.28 SIII +0.12 TV6-0.148

DIAGNOSTIC ACCURACY,

Sokolow-Lyon and Romhilt- Estes Criteria ---- Sensitivity- 10 to 30%

Specificity-85 to 95%

Cornal and Noncode regression method ---- Sensitivity- 35to50%

Specificity- 85to95%

MATERIALS AND METHODS

STUDY PLACE:

This study was conducted at HYPERTENSION CLINIC of MEDICINE DEPARTMENT in every Monday and Saturday in Government Stanley hospital during August 2004 to April 2005.

STUDY POPULATION:

This study is based on the analysis of the ECG of 800 patients who attended the hypertension clinic. It included 545 male patients and 255 female patients.

INCLUTION CRITERIA:

1) Patient who had established hypertension and who were in regular antihypertensive medication.
2) Only those patients with proved **Primary hypertension** were included in this study.
3) Of these, patients with consistent elevation of blood pressure over a period of **three weeks** only were taken up for ECG analysis.

EXCLUTION CRITERIA:

1. The patients with **secondary hypertension** and **diabetes mellitus** are excluded.
2. The patients with **ISOLATED SYSTOLIC HYPERTENSION** are excluded.
3. The patients with **NON SUSTAINED** hypertension are excluded.

METHOD:

-- Blood pressure was measured in all the **four limbs**, recumbent, sitting and standing postures were used.

--Routine urine analysis and blood for Urea, chlostereral, Electrolyte and Creatinine were done.

-- **Complete clinical examination** was done to exclude the possibility of secondary hypertension.

-- Standered 12 leads ECG was taken and the patient who were having ECG abnormality were followed up a week later with another ECG and only patients with persistent abnormality were taken for analysis.

THE RESULT, OBERVATION AND DISCUSSION

ECG CHANGE	NUMBER	PERCENTAGE
Normal ECG	498	62.25
Myocardial ischemia	76	9.5
Old myocardial infarction	24	3
Left ventricular hypertrophy Without Strain With Strain	 64 30	 8 3.75
Ventricular premature contraction	8	1
Left axis deviation	20	2.5
Conduction defect	**80**	**10**
RBBB	**14**	**1.75**
LBBB	**8**	**1**
LAFB	**58**	**7.25**
TOTAL	800	100

OBSERVATION

The ECG of 800 consecutive patients with persistent hypertension was analyzed for Conduction defect as described above.

94 cases have abnormal ECG's of which Left ventricular hypertrophy with and without strain formed the main abnormality in this study. Conduction disturbance in the form of RBBB, LBBB, LAFB were present in 10% of cases. Among which LAFB was the most common type of Conduction disturbance and it constitute7.25%.

Thirteen ECG's with LAFB also showed features of LVH with strain. Ten ECG'S showed LAFB had feature of Anterolateral myocardial Ischemia as well as one ECG with LAFB had LVH without strain.

Two EEG with LAFB showed evidence of old ASMI. Two ECG with LAFB showed LVH with evidence of diastolic overload, Eight ECG showed Ventricular premature Contracture.

Among the EEG with evidence of RBBB, six of them had Ventricular hypertrophy.

Ten ECG showing LBBB was not associated with any evidence of ischemia, Ventricular hypertrophy. NO case with LPFB was observed among the abnormal electrocardiogram.

INCIDENCE OF CONDUCTION DEFECTS IN MALES and FEMALES

TYPE	MALE	FEMALE
LAFB	40	18
LBBB	5	3
RBBB	9	5
TOTAL	54	26

INCIDENCE OF CONDUCTION DEFECT WITH INCREASING DIASTOILC BLOOD PRESSURE

TYPE	DBP< 140mmhg	DBP > 140mmhg
LAFB	9	35
RBBB	4	6
LBBB	2	3
TOTAL	15	44

DISCUSSION

From the results it is evident that conduction defects form one of the main ECG change in Essential Hypertension. Closely following are the incidence of Left ventricular Hypertrophy and Myocardial ischemia.

Eighteen out of eighty patients with conduction defect also showed evidence of Myocardial Ischemia. While fourteen out of Eighty patients with conduction defect showed evidence of Left ventricular hypertrophy. Nearly 50% of patients with conduction defect had other evidence of Hypertensive heart disease, which might have predisposed to the development of Inter ventricular conduction disturbance.

The sub endocardial fibrosis may be the reason for the involvement of conduction pathway in the thirty cases which showed no evidence of ventricular Hypertrophy or Ischemia.

When considering the relative incidence of various conduction defects in Male hypertensives and Female hypertensives, it is nearly 2:1 respectively for LAFB and LBBB. Where at it is 3:4 as RBBB was concerned. These facts indicate that male patients are more prone to develop cardiac complication of Hypertension, such as conduction disturbance.

When patients were arbitrarily divided into two groups depending on their diastolic blood pressure below or above 140 mm hg to find out if there is any relation of complications like conduction defects with reference to the weight of Diastolic blood pressure, it was found that patients showing LAFB, 35 were

having a BP more than 140mmhg while only 9 were having a BP of less than 140mmhg with a ratio of 4:1. Similarly the incidence of RBBB and LBBB were greater with patients having a DBP of more than 140 mm hg with a ratio of 4:3 and 2:1 respectively, suggesting that a high DBP over prolonged periods certainly leads to many cardiac complication (Myocardial ischemia secondary to LVH are also common with High blood pressure)

Though an objective study was made regarding the number of years of hypertension and the occurrence of conductive disturbances, no decisive inference could be taken, because many patients were aware of having hypertension only from the day they visited a doctor for some other complaint (hypertension was accidentally detected by them). While some patients even though , they had a few symptoms like headache and giddiness referable to hypertension, were having indigenous treatment or delayed consultation with a physician for an indefinite period. So the exact duration of hypertension in them could not be gauged.

Conduction disturbances as a part of cardiovascular complication of hypertension is proved once again in this study. Other studies like FRAMINGHAM study has shown that the incidence of Arrhythmias, Conduction defect and sudden death is considerably higher among hypertensives [40,42,43].

CHUNG in his text book on ECG states that hypertension is one of the commonest causes of conduction disturbances in the heart.

A review of Hypertension and LVH by FRANZ H. MASSERLF et al. In the cardiology clinics (NOV-1995) states that the increased incidence of

conduction disturbances in hypertensive is probably due to the increased fibrous tissue or altered collagen content.

MARTIN et al in their recent electrophysiological studies have demonstrated that conduction disturbances develop in hypertrophied ventricles in the presence of myocardial ischemia. Lot of studies has proved that presence of coronary ischemia with normal coronary arteries in hypertension. (CICULATION 1-18, 1998)

While describing about left ventricular hypertrophy as one of the causes of LAFB, GOLDMAN suggests, abnormal degree of left axis deviation is probably not due to the hypertrophied mass as such, but to associated sub endocardial fibrosis involving the LAFB.

GOPINATH et al in 1994 , during their 3 years follow up study of hypertension in Delhi recorded ECG of 871 patients have had abnormal ECG'S were exhibited by 307 patients (35.2%) of which 24 patients had Myocardial infarct,133(15.3%) patients were had ischemic ST-T changes , 54 patients (6.2%) had LVH and 96 (11%) had conduction disturbances and arrhythmias.

In our study the number of patients showing abnormal ECGs with old infarction, correlate very well with the above studies, while the incidences of conduction disturbance and arrhythmia were slightly lower—as opposed to 11%, the incidence of LVH was higher in our study group (11.75%)compared to WHO bulletin 1994:725 (6.2%).

SUMMARY

This study was conducted among 800 patients with hypertension attending Hypertension clinic at GOVERNMENT STANLEY HOSPITAL – CHENNAI from August 2004 to April 2005.

Standard 12 leads ECG was recorded in all patients and analyzed for the presence of intraventricular conduction defect like RBBB, LBBB, LAFB and LPFB ect. using the standard electrocardiographic criteria for such defects.

Our analysis of the ECGs showed the presence of abnormal ECGs in 37.75% of patients. Commonest abnormality is LVH (11.75%) followed by Conduction disturbance (10%), myocardial ischemia (9.5%), old Myocardial infarct (3%), Left axis deviation (2.5%), Ventricular premature contraction (1%). while 62.25% of patients had a normal ECG.

The result of our studies proves that hypertension is frequently associated with the development of conduction disturbances and increased severity of hypertension may be related to increased incidence of such defect. The identification of which may be helpful in defining high risk groups and start appropriate therapy to prevent complications and to reduce the morbidity and mortality.

CONCLUSION

1. This study proves that intraventricular conduction disturbance is one of the commonest electrocardiographic manifestations in Primary hypertention.
2. Left anterior fascicular block (7.25%) is the commonest one followed by Right (1.75%) and Left bundle branch block (1%).
3. With increasing diastolic BP conduction disturbances become more common.

BIBLIOGRAPHY

I) Braunwald's text book of cardiology

2) Hurst's text book of medicine

3) Harrison's text book of medicine

4) Cardiology clinics vol13 number-4 Nov-1995

5) American journal of cardiology 17, 1277:1991

6) American journal of Medicine 75 (sup 3A) 4, 1983

7) Journal of American college of cardiology 19:803, 1992

8) Goldman principals of clinical ECG

9) WHO bulletin 1994.725-20

10) Bargava-R.M.et al –Electrocardiographic changes in hypertension-Indian Heart journal 18:1966.

11) Bhaskar Reddy et al-Hypertensive heart disease the antiseptic 5:293, 1973.

12) Symposium on hypertension B.M.J 433:1973

13)Chyer Pand Kissane-Hypertension-American journal of Medicine 56:370,1974.

14) Evans. N-Electro Cardiogram in diagnosis of systemic hypertension-British heart journal 24:1, 1962

15) George C.F. et al-Value of routine ECG in hypertension patient –British Heart journal 34:618, 1972.

16) Leisman A.W.D-The ECG in hypertension-Quarterly Journal of Medicine 20:1, 1951.

17) Maurice Lev –Circulation 1972-pathological correlation.

18) De Wardener H.E Mac Gregor G.A.(1982)-The natriuretic hormone and essential hypertension Lancet1;1450-54.

19) Lewish Kuller MD et al-Therapy and Prevention of hypertension circulation jan 1989(American heart Association)

20) Page –Arterial hypertension in retrospective Res34:133-144, 1974.

21) Sino-atrial node dysfunction, atrio ventricular block and intra ventricular conductance disturbance, E.Rowland and F.Morgado,Europian heart journal, Volume 13, Supplement II, december1992.

22) Hypertension, European heart journal, Volume 15, Abstract Supplement, August 1992.

23) Clinical electrocardiography,B.L.Chia

24) Lipman,massie –Clinical Electrocardiography.

25) An introduction to Electrocardiography,Leo Schamroth.

26) Hiss RG,Lamb LE.Electrocardiograpic findings in 122,043. Circulation 1962:25:947.

27) Klein RC, Vera Z,DeMaria AN, et al. Electrocardiograpic diagnosis of left ventricular hypertrophy in the presence of left bundle dranch block. Am Heart J 1984:108:502-506.

28)Willems JL, R OBLES De Medina EO,Bernard R,et al.Criteria for intraventricular-conduction disturbances and preexcitation.Jam Coll Cardiol 1985;5:1261-1275.

29)Rotman M,Tribwasser JH.A clinical and follow-up study of right and left bundle branch block. Circulation 1975; 51:477-484.

30)Lesser RP, Haft JI, Friesber CK. Relationship of right bundle-branch block and n\marked left axis deviation to complete heart block and syncope .circulation 1968;47:429-437.

31)Frohlich ED, Apstein C, Chobanian AV, et al. The heart in hypertension. N Engl J Med 1992;327:998-1008.

32)DePuey EG, Guertler-Krawczynska E, Perkins JV, Robbins WL, Whelchel JD, Clements SD. Alterations in myocardial thallium-201 distribution in patients with chronic systemic hypertension undergoing single-photon emission computed tomography. Am J Cardiol 1988;62:234-238.

33)Houghton JL, Frank MJ, Carr AA, von Dohlen TW, Prisant LM. Relations among impaired coronary flow reserve, left ventricular hypertrophy and thallium perfusion defects in hypertensive patients without obstructive coronary artery disease. J Am Coll Cardiol 1990;15:43-51.

34)Schulman DS, Francis CK, Black HR, Wackers FJT. Thallium-201 stress imaging in hypertensive patients. Hypertension 1987;10:16-21.

35)Kannel WB, Gordon T, Offutt D. Left ventricular hypertrophy by electrocardiogram: prevalence, incidence, and mortality in the Framingham Study. Ann Intern Med 1969;71:89-105.

36)Mosterd A, D'Agostino RB, Silbershatz H, et al. Trends in the prevalence of hypertension, antihypertensive therapy, and left ventricular hypertrophy from 1950 to 1989. N Engl J Med 1999;340:1221-1227.

37)Sokolow M, Perloff D. The prognosis of essential hypertension treated conservatively. Circulation 1961;23:697-713.

38)Dunn FG, McLenachan J, Isles CG, et al. Left ventricular hypertrophy and mortality in hypertension: an analysis of data from the Glasgow Blood Pressure Clinic. J Hypertens 1990;8:775-782.

39)Kreger BE, Cupples LA, Kannel WB. The electrocardiogram in prediction of sudden death: Framingham Study experience. Am Heart J 1987;113:377-382.

40)Pringle SD, Dunn FG, Macfarlane PW, McKillop JH, Lorimer AR, Cobbe SM. Significance of ventricular arrhythmias in systemic hypertension with left ventricular hypertrophy. Am J Cardiol 1992;69:913-917.

41)Frohlich ED, Tarazi RC, Dustan HP. Clinical-physiologic correlations in the development of hypertensive heart disease. Circulation 1971;44:446-455.

42)Casale PN, Devereux RB, Milner M, et al. Value of echocardiographic measurement of left ventricular mass in predicting cardiovascular morbid events in hypertensive men. Ann Intern Med 1986;105:173-178.

43)Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 1990;322:1561-1566.

44)Frohlich ED, Apstein C, Chobanian AV, et al. The heart in hypertension. N Engl J Med 1992;327:998-1008. [Erratum, N Engl J Med 1992;327:1768.]

45)Levy D, Salomon M, D'Agostino RB, Belanger AJ, Kannel WB. Prognostic implications of baseline electrocardiographic features and their serial changes in subjects with left ventricular hypertrophy. Circulation 1994;90:1786-1793.

46)Berlowitz DR, Ash AS, Hickey AC, et al. *In*adequate management of blood pressure *in* a hypertensive population. N Engl J Med 1998;339:1957-1963. Pedram, A., Razandi, M., Aitkenhead, M., Levin, E. R. (2005). Estrogen Inhibits hypertension: the heart and hypertension. *Heart* 86: 467-475

48) Aguilera MT, de la Sierra A, Coca A, et al: Effect of alcohol abstinence on blood pressure: assessment by 24-hour ambulatory blood pressure monitoring. Hypertension 1999 Feb; 33(2): 653-7

49)Almendral J, Villacastin JP, Arenal A, et al: Evidence favoring the hypothesis that ventricular arrhythmias have prognostic significance in left ventricular hypertrophy secondary to systemic hypertension. Am J Cardiol 1995 Nov 2; 76(13): 60D-63D

50) Burke AP, Farb A, Liang YH, et al: Effect of hypertension and cardiac hypertrophy on coronary artery morphology in sudden cardiac death. Circulation 1996 Dec 15; 94(12): 3138-45.

51)Cabezas M, Comellas A, Ramon Gomez J, et al: [Comparison of the sensitivity and specificity of the electrocardiography criteria for left ventricular hypertrophy according to the methods of Romhilt-Estes, Sokolow-Lyon, Cornell and Rodriguez Padial]. Rev Esp Cardiol 1997 Jan; 50(1): 31-5.

52)Chobanian AV, Bakris GL, Black HR, et al: The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA 2003 May 21; 289(19): 2560-72.

53)Conlin PR, Chow D, Miller ER 3rd, et al: The effect of dietary patterns on blood pressure control in hypertensive patients: results from the Dietary Approaches to Stop Hypertension (DASH) trial. Am J Hypertens 2000 Sep; 13(9): 949-55.

54)Dahlof B: Left ventricular hypertrophy and angiotensin II antagonists. Am J Hypertens 2001 Feb; 14(2): 174-82.

55)Dahlof B, Pennert K, Hansson L: Reversal of left ventricular hypertrophy in hypertensive patients. A metaanalysis of 109 treatment studies. Am J Hypertens 1992 Feb; 5(2): 95-110.

56)Drayer JI, Weber MA, DeYoung JL: BP as a determinant of cardiac left ventricular muscle mass. Arch Intern Med 1983 Jan; 143(1): 90-2.

57)Fagard R, Pardaens K: Left ventricular diastolic function predicts outcome in uncomplicated hypertension. Am J Hypertens 2001 Jun; 14(6 Pt 1): 504-8.

58)Galderisi M, Petrocelli A, Alfieri A, et al: Impact of ambulatory blood pressure on left ventricular diastolic dysfunction in uncomplicated arterial systemic hypertension. Am J Cardiol 1996 Mar 15; 77(8): 597-601.

59)Ghali JK, Kadakia S, Cooper RS, Liao YL: Impact of left ventricular hypertrophy on ventricular arrhythmias in the absence of coronary artery disease. J Am Coll Cardiol 1991 May; 17(6): 1277-82.

60)Ghali JK, Liao Y, Simmons B, et al: The prognostic role of left ventricular hypertrophy in patients with or without coronary artery disease. Ann Intern Med 1992 Nov 15; 117(10): 831-6.

61) Gillman MW, Kannel WB, Belanger A, D'Agostino RB: Influence of heart rate on mortality among persons with hypertension: the Framingham Study. Am Heart J 1993 Apr; 125(4): 1148-54.

62)Gottdiener JS, Thurmann PA, Kenedi P, et al: Influence of the angiotensin II antagonist valsartan on left ventricular hypertrophy in patients with essential hypertension. Circulation 1999 Aug 10; 100(6): 685-8.

63) Greenberg B, Quinones MA, Koilpillai C, et al: Effects of long-term enalapril therapy on cardiac structure and function in patients with left ventricular

dysfunction. Results of the SOLVD echocardiography substudy. Circulation 1995 May 15; 91(10): 2573-81.

64)Harrap SB, Dominiczak AF, Fraser R, et al: Plasma angiotensin II, predisposition to hypertension, and left ventricular size in healthy young adults. Circulation 1996 Mar 15; 93(6): 1148-54.

65)Iriarte M, Murga N, Sagastagoitia D, et al: Congestive heart failure from left ventricular diastolic dysfunction in systemic hypertension. Am J Cardiol 1993 Feb 1; 71(4): 308-12.

66)Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. Arch Intern Med 1997 Nov 24; 157(21): 2413-46.

67) Jones DW, Hall JE: Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure and evidence from new hypertension trials. Hypertension 2004 Jan; 43(1): 1-3.

68) Kahn S, Frishman WH, Weissman S, et al: Left ventricular hypertrophy on electrocardiogram: prognostic implications from a 10-year cohort study of older

subjects: a report from the Bronx Longitudinal Aging Study. J Am Geriatr Soc 1996 May; 44(5): 524-9.

69)Kannel WB, Cobb J: Left ventricular hypertrophy and mortality--results from the Framingham Study. Cardiology 1992; 81(4-5): 291-8.

70)Kannel WB: Left ventricular hypertrophy as a risk factor: the Framingham experience. J Hypertens Suppl 1991 Dec; 9(2): S3-8; discussion S8-9.

71) Kaplan NM: Systemic hypertension: mechanisms and diagnosis. In: Braunwald E, ed. Heart disease. A Textbook of Cardiovascular Medicine. Philadelphia, Pa: WB Saunders; 1997: 807-22.

72)Konstam MA, Kronenberg MW, Rousseau MF, et al: Effects of the angiotensin converting enzyme inhibitor enalapril on the long-term progression of left ventricular dilatation in patients with asymptomatic systolic dysfunction. SOLVD (Studies of Left Ventricular Dysfunction) Investigators. Circulation 1993 Nov; 88(5 Pt 1): 2277-83.

73) Levy D, Salomon M, D'Agostino RB, et al: Prognostic implications of baseline electrocardiographic features and their serial changes in subjects with left ventricular hypertrophy. Circulation 1994 Oct; 90(4): 1786-93.

74) Levy D, Garrison RJ, Savage DD, et al: Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 1990 May 31; 322(22): 1561-6.

75)Liao Y, Cooper RS, Mensah GA, McGee DL: Left ventricular hypertrophy has a greater impact on survival in women than in men. Circulation 1995 Aug 15; 92(4): 805-10. .

76)Liebson PR, Grandits GA, Dianzumba S, et al: Comparison of five antihypertensive monotherapies and placebo for change in left ventricular mass in patients receiving nutritional- hygienic therapy in the Treatment of Mild Hypertension Study (TOMHS). Circulation 1995 Feb 1; 91(3): 698-706.

77)Liebson PR, Grandits G, Prineas R, et al: Echocardiographic correlates of left ventricular structure among 844 mildly hypertensive men and women in the Treatment of Mild Hypertension Study (TOMHS). Circulation 1993 Feb; 87(2): 476-86.

78)Mayet J, Chapman N, Shahi M, et al: The effects on cardiac arrhythmias of antihypertensive therapy causing regression of left ventricular hypertrophy. Am J Hypertens 1997 Jun; 10(6): 611-8.

79)Mayet J, Ariff B, Wasan B, et al: Improvement in midwall myocardial shortening with regression of left ventricular hypertrophy. Hypertension 2000 Nov; 36(5): 755-9.

80)Mensah GA, Francis CK, Crawford MJ, et al: Complications of hypertension: the heart. Cardiology 2000; 1: 3-4, 1-12.

81)Messerli FH, Grodzicki T: Hypertension, left ventricular hypertrophy, ventricular arrhythmias and sudden death. Eur Heart J 1992 Sep; 13 Suppl D: 66-9.

82)Pitt B: Regression of left ventricular hypertrophy in patients with hypertension: blockade of the renin-angiotensin-aldosterone system. Circulation 1998 Nov 10; 98(19): 1987-9.

.

PROFORMA

Name : Religion

Age :

Sex : Address :

Occupation :

Income :

Age of onset of HT :

Duration of HT :

Treatment History : Diet/Oral drugs

Dietary Habits : Veg /Non-Veg/ Mixed

Smoker : Yes/No Duration:

Alcoholic : Yes/No Duration:

Amount:

Family History : Hypertension Yes/No

Diabetes Yes/No

CAD Yes/No

Hyperlipidemia Yes/No

H/O Diabetes Yes/No Duration : Drugs;

IHD Angina/Infarction Yes/No

Hyperlipidemia Yes/No

CLINICAL EXAMINATION

Hight: BMI:

Weight: Sarface area:

Pulse: Peripheral arteries:

BP: Supine: Right UL: Left UL:

Standing: Right LL: Left LL:

CVS: S1 S2 Murmur

RS :

ABDOMEN: Mass Liver Spleen ECG:

Rate:

Rhythm:

Axis:

P wave:

PR interval:

QRS duration:

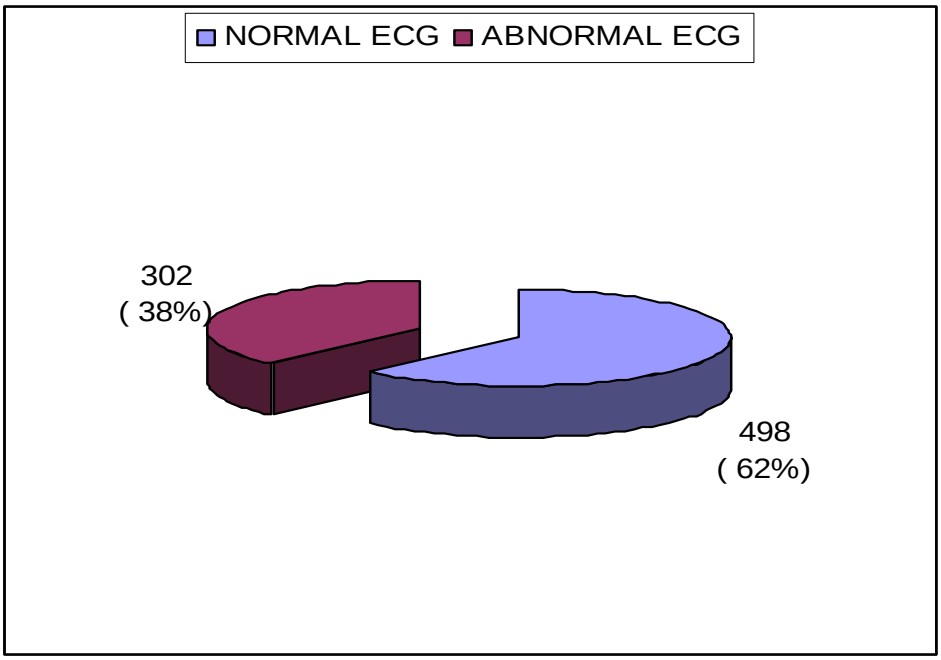
QRS morphology:

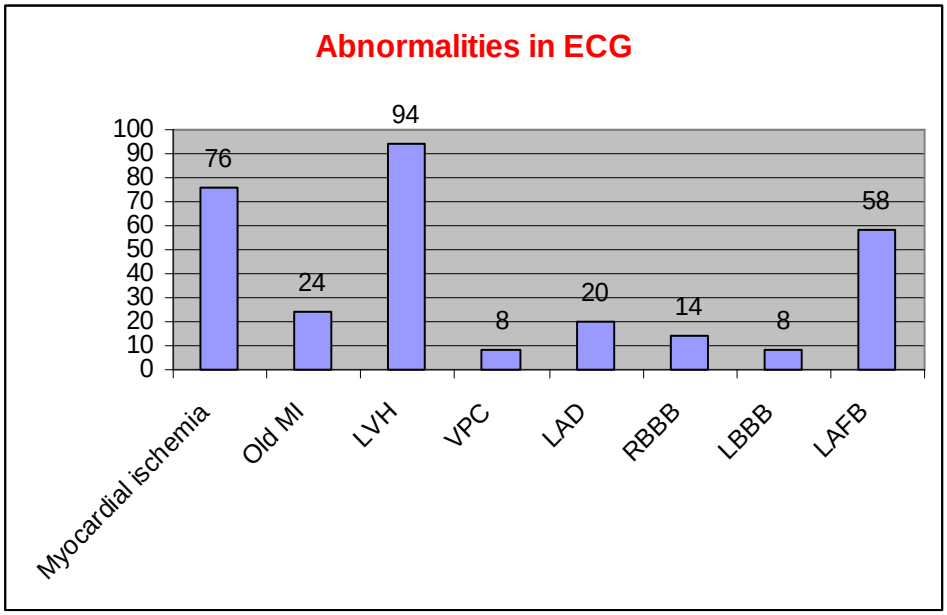
LVH by ROMHILT-ESTES POINT SCORE SYSTEM

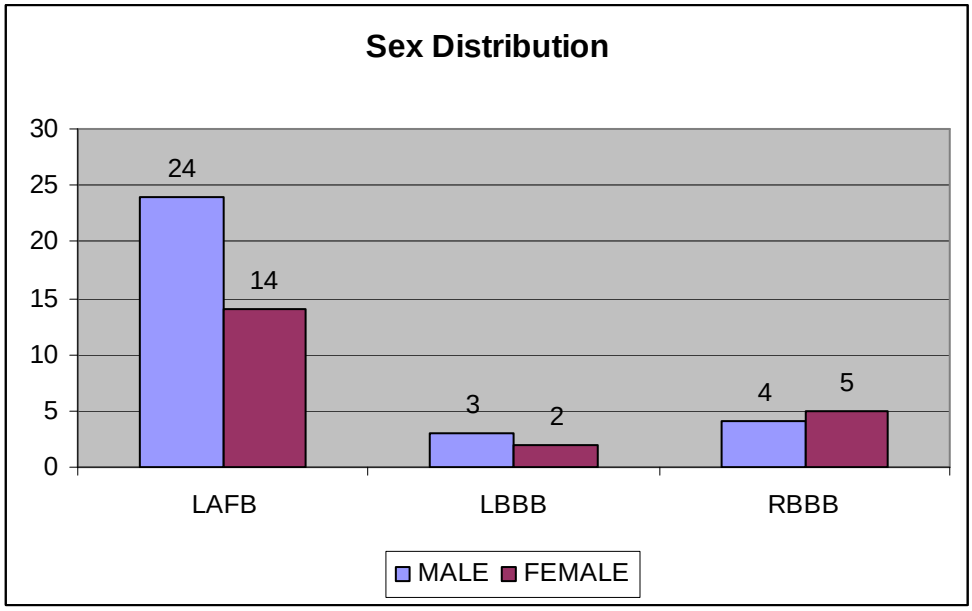
S.NO	AGE	SEX	B.P	No of years of Hypertension	RHYTHM	RATE	QRS	VAT	QRS Axis	QRS Configuration
1	60	M	190/120	7	NSR	86	0.08	0.03	+30	qR in I, aVL, rS in II, III, aVF
2	70	M	166/120	12	NSR	94	0.07	0.04	+40	qR in I, aVL, rS in II, III, aVF
3	56	M	180/148	6	NSR	78	0.08	0.03	+15	qR in I, aVL, rS in II, III, aVF
4	67	F	200/150	15	NSR	88	0.08	0.04	+60	qR in I, aVL, rS in II, III, aVF
5	66	M	180/140	8	NSR	84	0.08	0.04	-15	qR in I, aVL, rS in II, III, aVF
6	58	M	160/110	11	NSR	86	0.08	0.04	+60	qR in I, aVL, rS in II, III, aVF
7	49	M	194/134	3	NSR	98	0.08	0.04	0	qR in I, aVL, rS in II, III, aVF
8	62	M	200/140	4	NSR	86	0.08	0.04	-35	qR in I, aVL, rS in II, III, aVF
9	68	M	184/124	10	NSR	102	0.08	0.04	+30	qR in I, aVL, rS in II, III, aVF
10	58	M	168/144	5	NSR	84	0.08	0.04	+5	qR in I, aVL, rS in II, III, aVF
11	65	F	174/122	3	NSR	82	0.07	0.03	+30	qR in I, aVL, rS in II, III, aVF
12	61	M	158/110	6	NSR	80	0.08	0.04	-45	qR in I, aVL, rS in II, III, aVF
13	60	M	160/132	12	NSR	78	0.08	0.04	-30	qR in I, aVL, rS in II, III, aVF
14	55	M	168/112	6	NSR	82	0.08	0.04	+30	qR in I, aVL, rS in II, III, aVF
15	48	M	200/164	8	NSR	84	0.07	0.03	+20	qR in I, aVL, rS in II, III, aVF
16	70	M	180/136	4	NSR	92	0.08	0.04	+15	qR in I, aVL, rS in II, III, aVF
17	56	F	174/142	11	NSR	74	0.08	0.04	-45	qR in I, aVL, rS in II, III, aVF
18	58	M	180/144	5	NSR	82	0.08	0.04	+15	qR in I, aVL, rS in II, III, aVF
19	63	F	176/120	9	NSR	80	0.08	0.03	-30	qR in I, aVL, rS in II, III, aVF
20	69	F	184/134	13	NSR	76	0.07	0.04	-45	qR in I, aVL, rS in II, III, aVF
21	59	M	200/146	11	NSR	70	0.08	0.04	-35	qR in I, aVL, rS in II, III, aVF
22	62	M	178/134	7	NSR	78	0.08	0.04	-30	qR in I, aVL, rS in II, III, aVF
23	66	M	180/154	9	NSR	92	0.08	0.03	- 45	qR in I, aVL, rS in II, III, aVF
24	64	M	196/128	15	NSR	102	0.08	0.04	-5	qR in I, aVL, rS in II, III, aVF
25	60	M	182/136	20	NSR	88	0.07	0.04	0	qR in I, aVL, rS in II, III, aVF
26	50	M	176/122	9	NSR	80	0.08	0.03	+60	qR in I, aVL, rS in II, III, aVF
27	58	M	168/134	11	NSR	108	0.08	0.04	+30	qR in I, aVL, rS in II, III, aVF
28	65	M	156/112	12	NSR	78	0.08	0.04	+65	qR in I, aVL, rS in II, III, aVF

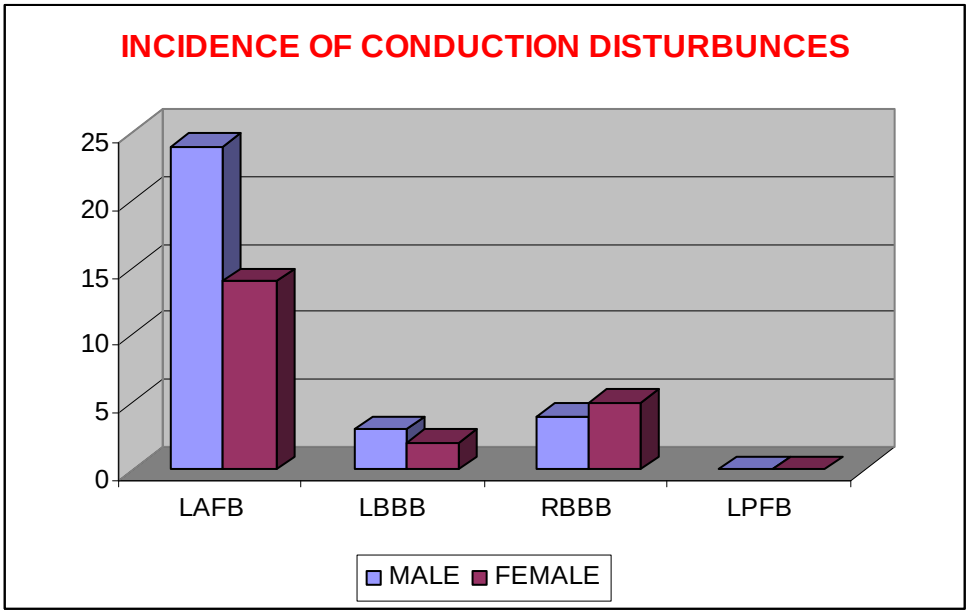
C.NO	AGE	SEX	B.P	No of years of Hypertension	RHYTHM	RATE	QRS	VAT	QRS Axis	QRS Configuration
29	57	F	180/140	7	NSR	86	0.07	0.04	+30	qR in I, aVL, rS in II, III, aVF
30	63	M	192/154	4	NSR	112	0.08	0.04	-45	qR in I, aVL, rS in II, III, aVF
31	61	M	176/144	11	NSR	78	0.06	0.02	-30	qR in I, aVL, rS in II, III, aVF
32	67	F	200/134	8	NSR	88	0.08	0.04	-55	qR in I, aVL, rS in II, III, aVF
33	59	M	166/140	5	NSR	92	0.08	0.03	-45	qR in I, aVL, rS in II, III, aVF
34	68	M	186/150	6	NSR	74	0.08	0.04	-30	qR in I, aVL, rS in II, III, aVF
35	50	M	190/160	16	NSR	100	0.08	0.03	+20	qR in I, aVL, rS in II, III, aVF
36	52	M	184/136	9	NSR	94	0.08	0.04	-30	qR in I, aVL, rS in II, III, aVF
37	58	M	178/154	14	NSR	82	0.08	0.04	+20	qR in I, aVL, rS in II, III, aVF
38	49	M	186/150	11	NSR	70	0.08	0.04	+55	qR in I, aVL, rS in II, III, aVF
39	72	M	196/154	10	NSR	86	0.07	0.04	+15	qR in I, aVL, rS in II, III, aVF
40	55	F	200/160	6	NSR	82	0.08	0.04	-15	qR in I, aVL, rS in II, III, aVF
41	56	M	194/166	8	NSR	80	0.08	0.03	-45	qR in I, aVL, rS in II, III, aVF
42	65	M	182/142	9	NSR	84	0.08	0.03	-10	qR in I, aVL, rS in II, III, aVF
43	48	M	168/140	13	NSR	78	0.07	0.04	-45	qR in I, aVL, rS in II, III, aVF
44	57	M	188/150	18	NSR	74	0.08	0.03	+60	qR in I, aVL, rS in II, III, aVF
45	68	M	212/146	9	NSR	106	0.07	0.04	-30	qR in I, aVL, rS in II, III, aVF
46	69	F	196/160	10	NSR	86	0.08	0.03	+65	qR in I, aVL, rS in II, III, aVF
47	65	M	202/158	19	NSR	90	0.08	0.04	+40	qR in I, aVL, rS in II, III, aVF
48	64	M	166/124	16	NSR	70	0.08	0.03	-45	qR in I, aVL, rS in II, III, aVF
49	63	M	186/142	12	NSR	94	0.07	0.04	+15	qR in I, aVL, rS in II, III, aVF
50	60	M	196/134	17	NSR	82	0.08	0.03	-35	qR in I, aVL, rS in II, III, aVF
51	61	M	180/144	10	NSR	104	0.08	0.04	+15	qR in I, aVL, rS in II, III, aVF
52	64	M	188/138	15	NSR	92	0.08	0.04	-30	qR in I, aVL, rS in II, III, aVF
53	61	F	204/164	6	NSR	78	0.08	0.03	+20	qR in I, aVL, rS in II, III, aVF
54	59	M	196/144	4	NSR	94	0.08	0.04	+45	qR in I, aVL, rS in II, III, aVF
55	72	M	190/160	10	NSR	72	0.08	0.03	-30	qR in I, aVL, rS in II, III, aVF
56	68	M	176/132	5	NSR	78	0.08	0.04	-45	qR in I, aVL, rS in II, III, aVF

C.NO	AGE	SEX	B.P	NO of yearsof Hypertension	RHYTHM	RATE	QRS	VAT	QRS Axis	QRS Configuration
57	60	M	188/146	6	NSR	76	0.08	0.03	+55	qR in I, aVL, rS in II, III, aVF
58	61	M	206/168	5	NSR	80	0.07	0.04	+20	qR in I, aVL, rS in II, III, aVF
59	63	M	210/168	8	NSR	92	0.08	0.03	+60	RR in VI V2 wides in V5 V6 I aVL
60	59	F	204/180	9	NSR	72	0.08	0.04	+10	rSR in V1 V2wide sin I V5 V6
61	58	M	198/178	12	NSR	68	0.08	0.03	+15	rSR in V1wide s in I II V456
62	60	M	180/140	17	NSR	72	0.07	0.04	-15	RR in VI V2 wide s in V5 V6 I aVL
63	69	M	178/144	8	NSR	70	0.08	0.03	+45	rSR in V1 V2 wide s in I V5 V6
64	52	F	194/148	3	NSR	104	0.08	0.04	+5	rSR in V1 wide s in I II V456
65	70	F	176/146	6	NSR	86	0.08	0.03	+40	rSR in V1 V2 wide s in I V5 V6
66	54	M	210/166	11	NSR	82	0.08	0.04	-30	RR in VI V2 wide s in V5 V6 I aVL
67	62	M	202/166	18	NSR	76	0.08	0.04	+65	rSR in V1 V2 wide s in I V5 V6
68	49	M	168/138	10	NSR	88	0.08	0.03	+40	rSR in V1 V2 wide s in I V5 V6
69	65	M	194/156	8	NSR	72	0.08	0.04	-25	RR in VI V2 wide s in V5 V6 I aVL
70	57	M	208/166	9	NSR	70	0.08	0.04	+15	rSR in V1 V2wide sin I V5 V6
71	45	M	164/128	4	NSR	92	0.07	0.04	+45	rSR in V1 V2wide sin I V5 V6
72	72	M	180/150	16	NSR	84	0.08	0.03	+60	RR in VI V2 wide s in V5 V6 I aVL
73	64	F	186/148	6	NSR	78	0.08	0.04	+20	Slurred R in V5,V6 wide s inV1
74	60	M	210/168	3	NSR	70	0.08	0.04	+35	RR in V6 wide s in V1,V2
75	59	M	188/160	7	NSR	82	0.08	0.03	+30	M pattern in V5,V6 wide s in VI
76	52	M	166/138	10	NSR	80	0.08	0.04	-45	Slurred R in V5,V6 wide s inV1
77	59	F	192/164	3	NSR	70	0.08	0.04	-30	RR in V6 wide s in V1,V2
78	66	M	212/168	16	NSR	88	0.08	0.04	-60	Slurred R in V5,V6 wide s inV1
79	50	M	174/146	12	NSR	74	0.08	0.03	+65	RR in V6 wide s in V1,V2
80	48	M	190/144	5	NSR	92	0.08	0.04	+35	M pattern in V5,V6 wide s in VI

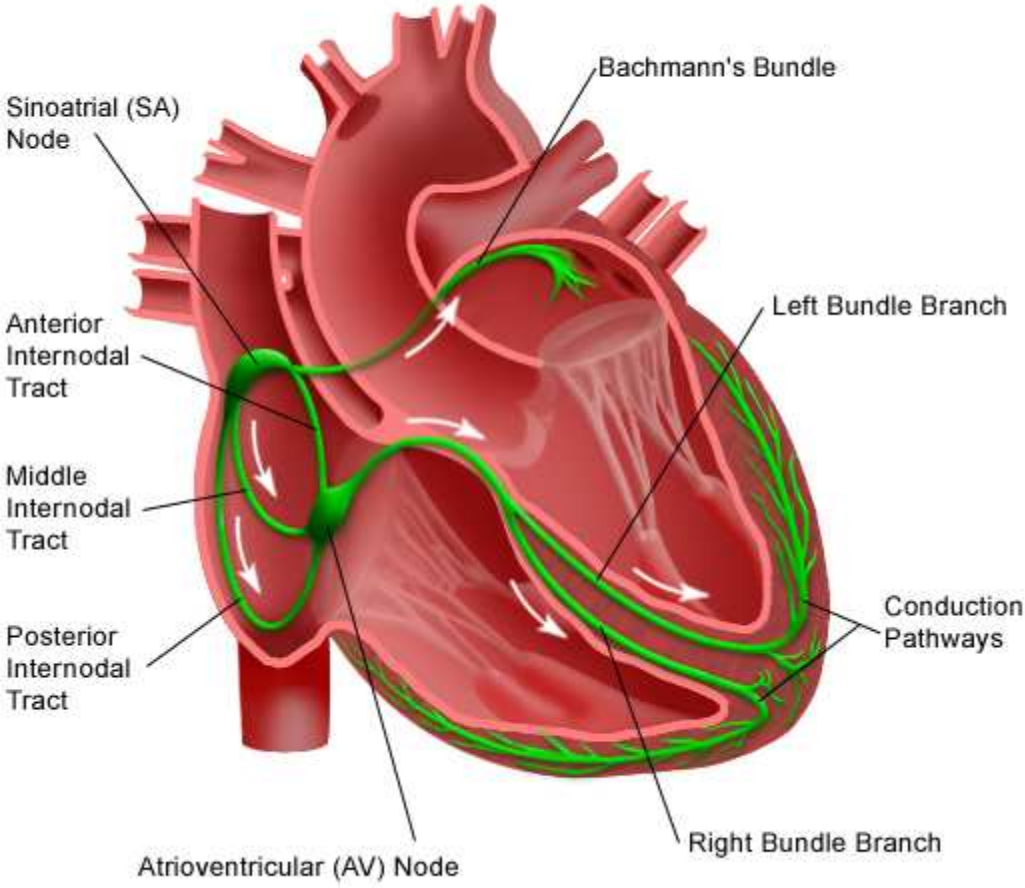

NORMAL ECG
ABNORMAL ECG
302
(38%)
498
(62%)


Abnormalities in ECG
100
90
80
70
60
50
40
30
20
10
0
76
24
94
8
20
14
8
58
Myocardial ischemia
Old MI
LVH
VPC
LAD
RBBB
LBBB
LAFB

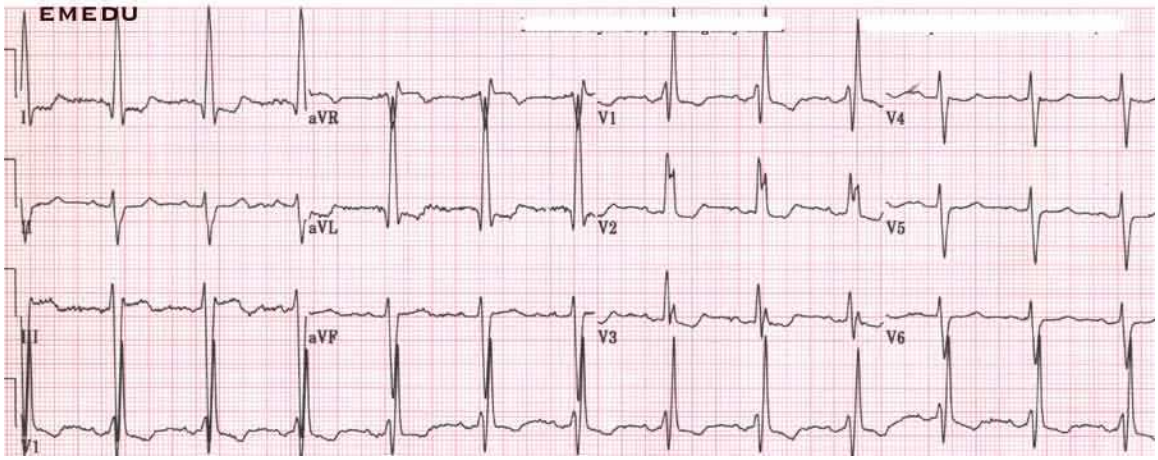

Sex Distribution
30
25
20
15
10
5
0
24
14
3
2
4
5
LAFB
LBBB
RBBB
MALE
FEMALE


INCIDENCE OF CONDUCTION DISTURBUNCES
25
20
15
10
5
0
LAFB
LBBB
RBBB
LPFB
MALE
FEMALE

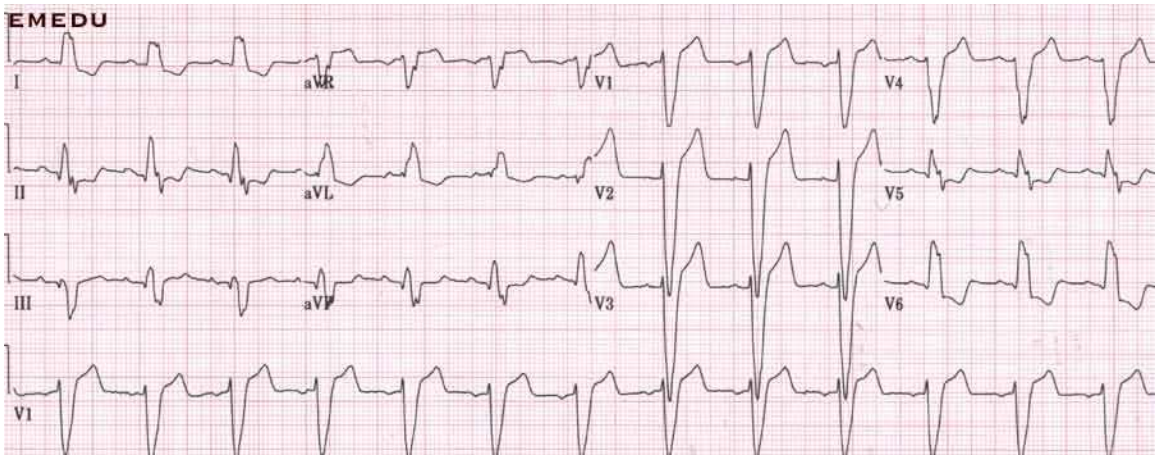
Electrical System of the Heart



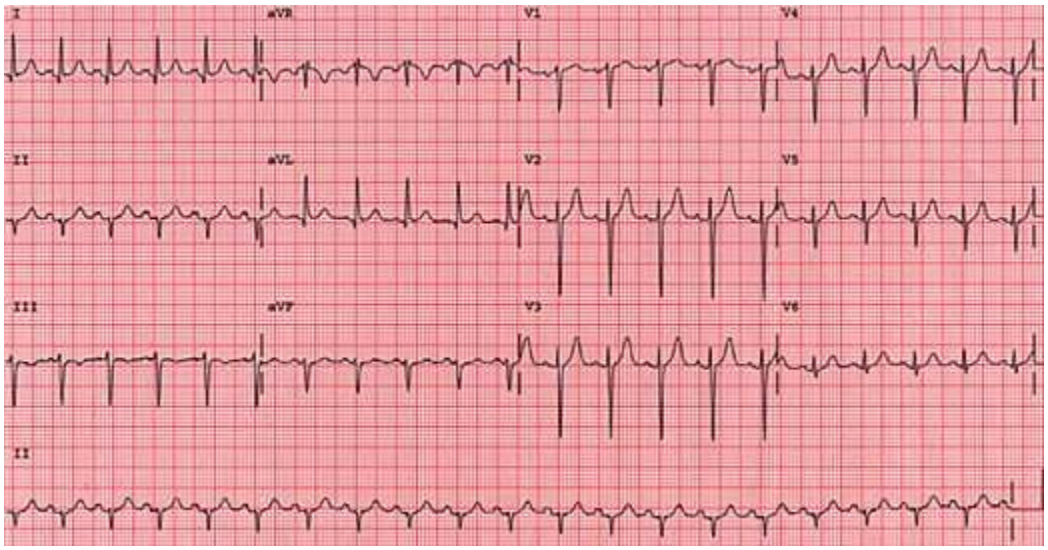
RBBB


EMEDU
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1

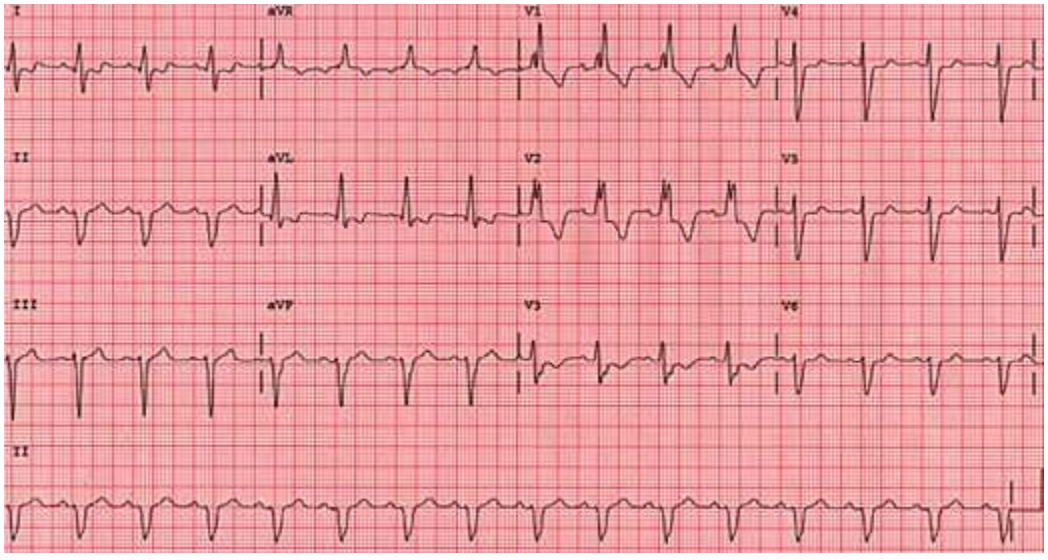
LBBB


EMEDU
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1

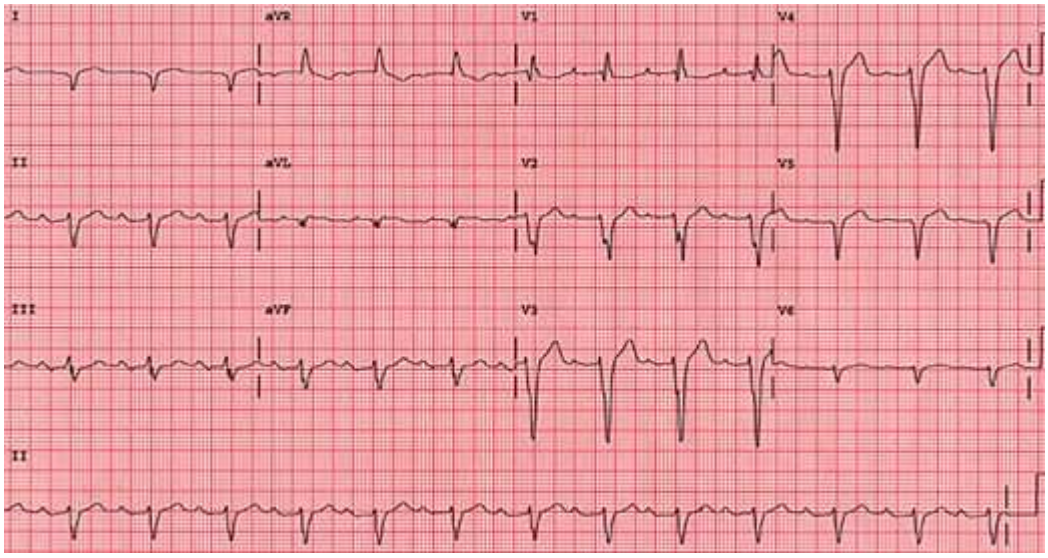
LAFB



RBBB with LAFB



LPFB



LEFT VENTRICULAR HYPERTROPHY